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12. THE ROLE OF NATIONAL POISON CONTROL CENTER IN ORGANISATION AND MANAGEMENT OF MASS AMMONIA ACCIDENT

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INTRODUCTION

Despite the frequent occurrence of incidents involving hazardous materials, organisation and management still pose some specific problems in chemical disasters. Most hospitals with emergency care are not fully prepared to handle contaminated or poisoned patients, which may have important impact on the outcome of poisoning (1). Following a hazardous chemical accident, providing early effective care to victims depends on the nature of the accident, the number of victims affected, the availability of medical care, the coordination of rescue teams and medical treatment. Poison Control Centres must have a crucial role in all phases of the disaster management.

METHODS

The objective of this paper is to review the role of the National Poison Control Centre (NPCC) in organisation and management of mass ammonia poisoning. It is an account of team work and experience in medical management of poisoning which was applied for the first time in such a major accident.

RESULTS

The accident happened on May 27th 1998 at 14:00 hrs in Belgrade's suburb Borca, when a road tanker with 5 tones of ammonia exploded. A cloud of ammonia gas spread over the vast area causing mass exposition of local residents and workers of "Lika system", a company located nearby. A man who was standing beside the truck was killed in the explosion. The Units of Urgent Medical Aid and Police Department were the first on the spot and before contacting NPCC, which is why it did not participate in the first phase of organisation of medical management. The duty physician in NPCC was informed at 14:40 hrs of the accident. He suggested that majority of victims should be transported to the Centre. Considering the fact that it was still the working hour, none of the staff left the hospital. A previously prepared plan regarding chain of command with triage physicians, a nurse in charge and the emergency staff call-up plan was put into motion. Additional hospital beds were provided by discharging from the Clinic of Toxicology and two other clinics of Military Medical Academy the patients who were hospitalised for various diagnostic interventions. Those patients who demanded further treatment were transferred to the Military Medical Centre. Additional supplies of drugs and other materials were provided. The patients started to arrive one hour after the accident and untill 22:00 hrs 98 patients were brought in. The Emergency Centre was not used for triage process because the number of patients was significant and they could overload and interfere with the care of severely poisoned patients. The entry for rapid access to triage and a separate, for that occasion specially formed area, near the entrance, for refreshment, observation and treatment of mild poisoning, was designated. Severely poisoned patients with vital threat were hospitalised immediately at the Clinic of Toxicology and Clinical Pharmacology. After admitting them, decontamination and copious eyes washing with isotonic saline were performed. During the next two days 45 patients with similar complaints reported to the Centre, so that total number of treated

patients with exposition or poisoning was 143, of which 54 were hospitalised. After first examination and triage, all the patients received a single dose of methylprednisolone (80 mg) *im* or *iv*. After six hours of observation 46 patients with mild poisoning or exposition only were discharged from the hospital. They remained under control during the period of one month on the outpatient basis. According to the severity of poisoning, hospitalised patients were divided into groups: mild (22 patients), moderate (13 patients) and severe poisoning (19 patients).

The majority of patients with mild poisoning had signs and symptoms of mild irritation of the upper respiratory tract (coughing, breathlessness, mild dyspnoea, mild bronchospasm, conjunctival hyperaemia). Those with moderate poisoning had prolonged coughing, chest tightness, carbonaceous sputum, wheezing and hypoxaemia requiring extra oxygen. Five of those patients had normal chest radiography and the rest bronchovascular markings. One of them had pneumonia. In patients with severe poisoning manifest respiratory insufficiency (due to severe bronchospasm, laryngeal obstruction due to pulmonary oedema, pneumonia), severe burns of eyes and skin were confirmed. Nine of them developed pulmonary oedema 6 hrs post exposure. In one of them oedema was a consequence of cardiac failure. Three patients had acute respiratory distress syndrome (ARDS) on admission and of those 2 were intubated and placed on mechanical ventilation (MV) with positive end-expiratory pressure. One of them deceased on the 6th day. Autopsy revealed severe haemorrhagic pulmonary oedema, bronchial mucosa destruction and multiple capillary thrombi. Second patient on MV developed pneumonia and pulmonary thromboembolism. He was hospitalised during the next 60 days and when discharged he still had restrictive-obstructive disturbances and mild hypoxaemia. His follow-up confirmed the development of pulmonary fibrosis.

Treatment included support of airway patency, frequent airway suction of bronchial and pulmonary secretion, application of humidified oxygen, intravenous fluids, bronchodilators, antibiotics and corticosteroids. For those with severe poisoning high doses of methylprednisolone (1.5-2.0 g) were administered in order to prevent the development of pulmonary oedema. They were reduced during next 72 hrs as the risk of oedema diminished. All patients but one survived. Hospitalization lasted for 2-60 days. Longterm follow-up confirmed sequelae in two patients. One of them had bronchial hyperreactivity and pulmonary fibrosis and other patient had chronic obstructive bronchitis and cataract (Table 1).

DISCUSSION

Since the early 1980s several chemical disasters involving release of toxic substances and mass poisonings have focused the attention of different services on enhancing safety in processing, handling and storage of dangerous chemicals and other hazardous industrial materials. The terrorist sarin attack in Tokyo subway in March 1995 showed that using the highly toxic agents by terrorist is feasible now and indicated the need for careful planning of organisation and management of accidents (2). Medical management of major chemical accidents requires a close collaboration between rescuers (on site and in hospital) and the Poison Control Centre, which toxicological databases and risk assesment software are integrated in management to support information sharing. According to the 1995 World Disaster Report, chemical accidents that put general public and environment into a serious danger rank 10th, just after epidemics (3). Close communication with the public, emergency care facilities, other authorities and the media play a key role in each step. In 1985 Major Accident Reporting System was established by the Comission of the European Community

(4). There are no published data about chemical accidents in Yugoslavia and this accident with mass poisoning is the first that we are aware of.

This paper explains how the National Poison Control Centre responded to such a situation from the viewpoint of accident management, the problems encountered and how they were solved. The NPCC participated actively for the first time in massive ammonia accident. Ammonia, a common industrial and household chemical, has a characteristic odour, which is a warning of exposure. After the accident the, first on the scene were units of Urgent Medical Aid and Police Department and they transported the majority of victims to NPCC. Thinking that ammonia is not a chemical with serious potential for contamination, as chemical carcinogens and pesticides are, they were not properly equipped so several rescuers were poisoned, but fortunately mildly. After first triage on spot, patients were transported to NPCC, where final triage was accomplished.

Clinical presentation of ammonia poisoning is characteristic, with respiratory irritation ranging from mild to severe pulmonary disturbances including ARDS. The mechanism of poisoning is explained by high water solubility of ammonia, which produces ammonium hydroxide that, along with excessive local heating caused by this chemical reaction, lead to mucosal destruction and alkaline burns. Contact with skin can cause burns and vesication, irritation of eyes, conjunctivitis and pharyngitis. It is absorbed in the upper respiratory tract, but exposure to highly concentrated gas or prolonged exposure may cause tracheobronchial or pulmonary inflammation. Exposure to high concentration of any irritant gas may produce hypoxaemia analogous to that seen with exposure to the simple asphyxiants.

General approach to managing patients with ammonia exposure is support of airway patency and supportive care is the mainstay of therapy. Supplemental oxygen, bronchodilators and airway suctioning should be used if clinically indicated. It is important to reduce the inspired concentration of oxygen to below 50% as rapidly as possible, since patients with ammonia poisoning may be even more susceptible to oxygen toxicity, due to the depletion of endogenous antioxidant system. Early corticosteroid therapy designed to reduce the host inflammatory response provides little benefit to patients with ARDS, but it may reduce the late fibroproliferative phase. The majority of our patients with mild and moderate poisoning were exposed to ammonia gas for a few minutes. After treatment they fully recovered and none of them had sequelae. On the contrary, patients presenting with signs of severe poisoning and ARDS, had prolonged clinical course and developed sequelae. Distinction could be made even in this group of patients, in which early onset of pulmonary oedema indicated the less severe form of poisoning and faster recovery.

All the victims were treated in NPCC, which might not be possible in the future, though statistics shows that many hospitals are not fully prepared to treat such patients. The survey of hospital-based facilities providing emergency care in the state of Washington, showed that only 44% of them reported the ability to receive any chemically exposed patient, while 70% had protocols for handling facility contamination and possible evacuation (1,6,7). However, regional medical management of chemical accidents with close cooperation with PCC is recommended because epidemiologic, laboratory and toxicological skills are needed immediately to evaluate and advise on chemical accident may not be available locally and delay an adequate medical response.

Our National Poison Control Centre consists of Clinic of Toxicology and Clinical Pharmacology, Institute of Toxicology and Pharmacology (Department of Analytical Toxicology, Department of Experimental Toxicology and Pharmacology, Toxicological Information Department) and Mobile Toxicological Unit. It has ten clinical toxicologists who treated these hundred patients successfully during several hours. It also provided authorities and mass media with all the necessary information on ammonia during all that

time and in that way the psychosociological response to chemical disaster with mass psychogenic illness was avoided. This indicates that NPCC was capable to deal with major medical problem and this experience will be of great help in dealing with possible chemical terrorist attack.

SUMMARY

National Poison Control Centre participated actively in organisation and medical management of mass ammonia accident. The incident showed high quality of treatment in such emergency care facility with multispecialised medical teams. It also indicates that NPCC should have a leading role in coordination of chemical disaster planning, teaching and medical training, information support, incident recording and liesons between the regional medical institutions and the government authorities.

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KEY WORDS

Ammonia, poisoning, chemical disaster, organization and medical management

TABLE

Table 1. A degree of poisoning, acute complications and poisoning sequelae

Degree	N pts	complication N	sequelae N
Mild	22	pneumonia 2	
moderate	13	pneumonia 1	
Severe	19	pneumonia 2, ARDS 9, ACF 1, PTE 1	p. fibrosis 1, b. hypersensitivity 1, cataract 1, COPD 1